United States Department of Agriculture

Animal and Plant Health Inspection Service

Program Aid 1576

Keeping America Free From Foreign Animal Diseases

Afican Horse Sickness

Volume 1

Guidelines for Using This Package

This binder contains an integrated suite of educational materials about African horse sickness. The package can be used in a formal training setting, where a presenter will show the video tape and narrate the slide show using this black-and-white brochure as the script. Or the materials can be used in a self-study program with the reader progressing at his or her own pace.

Within this brochure, readers will notice that certain paragraphs are preceded by a number. These numbers correlate to the slide set. For example, the African horse sickness slides are all marked "HS" at the top of each plastic slide frame and numbered sequentially from 1 to 46.

If you remove the slides from their protective clear-plastic sleeve (for example, to put them into a carousel for group viewing), please be sure to reposition them in the correct numeric order for the benefit of future users.

This shrink-wrapped suite includes one general and one scientific video tape on African horse sickness, a separate slide set on this disease, and the brochure you are reading now. If your package is incomplete, please contact the following office for replacement materials:

U.S. Department of Agriculture
Animal and Plant Health Inspection Service
Veterinary Services, Emergency Programs
4700 River Road, Unit 41
Riverdale, MD 20737–1231

Instructional packages on other diseases are also available and may be requested by writing to the above address. Titles include
Program Aid 1577 African Swine Fever
Program Aid 1578 Contagious Bovine Pleuropneumonia
Program Aid 1579 Lumpy Skin Disease, Sheep Pox, Goat Pox
Program Aid 1580 Malignant Catarrhal Fever
Program Aid 1581 Rinderpest and Peste des Petits Ruminants
Program Aid 1582 Vesicular Diseases

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1 African Horse Sickness

Definition

2 African horse sickness (AHS) is an acute or subacute, arthropod-borne, noncontagious viral disease of horses and other equidae that is often fatal. It is characterized by fever and by edema of the subcutaneous tissue and lungs.

Etiology

3 AHS is caused by a 68–70-nm double-stranded RNA virus in the family Reoviridae, genus *Orbivirus.* The AHS virus (AHSV) is very similar to the virus that causes bluetongue.

There are nine serotypes of AHSV.

The virus can be inactivated by repeated freezing and thawing and by treatment with acetic acid (inactivated at a pH 6.3 or lower), remaining for 2 weeks at 37 °C, and being placed for 5 minutes at 70 °C.

In OCG (5 g of potassium oxalate, 5 g of carbolic acid [phenol], 500 mL of water, and 500 mL of glycerine), AHSV is stable for more than 20 years at 4 °C.

Effective Disinfectants

4 Acetic acid (2 percent), iodophore disinfectants, and chlorine dioxide disinfectants are all effective.

History

5 AHS appears to be truly an African disease: it was not recognized before settlers from Europe took horses to South Africa in the middle of the 17th century. Since that time, major outbreaks have occurred in southern Africa about every 20 years. Serotypes 1–3 and 5–8 have been confined mainly to Africa. In 1959, type 9 appeared in Israel, Iran, Pakistan, and Afghanistan. In 1960, type 9 reappeared in these countries and spread to India, Turkey, Iraq, Syria, Lebanon, Jordan, and Cyprus. In 1961, the same strain reappeared in India, Pakistan, Turkey, Iran, Jordan, and Iraq. During this outbreak, as many as 300,000 animals may have died. In 1965–66, AHS

occurred in North African countries along the Mediterranean Sea and in Spain. Type 4 occurred in Spain in 1987–90 and in Morocco in the early 1990's.

Host Range

6 The hosts in order of decreasing severity of disease are horses, mules, donkeys, and zebras.

Horses and mules have the highest mortality, donkeys have a lower mortality, and African donkeys have a subclinical infection.

Zebras have a subclinical infection. They are believed to play a role in the persistence of the virus in Africa because zebras can be viremic for up to 6 weeks. But they are probably not the reservoir, for they eventually clear the viremia.

Dogs can become infected by eating AHSV-infected meat. According to 1 report, 31 out of 35 dogs became sick and 7 died of AHS after being fed infected horse meat. In the 1987–90 outbreak in Spain, dogs that did not eat infected meat were found to be seropositive, suggesting infection by arthropod bites.

Most laboratory animals (with the exception of rabbits) are susceptible by parenteral inoculation. Intracerebral inoculation of baby mice is the preferred method of isolating the virus.

There is no evidence that humans can be infected by field strains of AHSV. However, intranasal exposure to neurotrophic vaccine strains has caused encephalitis and retinitis in humans.

Geographic Distribution

7 The disease is endemic in equatorial, eastern, and southern Africa, from which it regularly spreads south and periodically north either along the Nile River or along the west coast of Africa.

Transmission

8

AHSV is spread by *Culicoides* spp. (midges or "no-see-em's") and clinically by the transfer of blood. *Culicoides* spp. are biological vectors because AHSV can replicate in the midge. In the United States, *Culicoides* that can transmit bluetongue virus most likely can transmit AHSV. *Culicoides* are most active at sunset and at about sunrise.

AHS is noncontagious, but the horse is an amplifier of AHSV and source of virus for arthropods. Arthropods other than the *Culicoides* (e.g., biting flies and mosquitoes) may spread the virus as mechanical vectors.

9

Culicoides feeding on a person's arm.

Epidemiology

10

In temperate climates, AHS outbreaks appear at the beginning of the warm season when *Culicoides* vectors appear and disappear 9 days after frost kills the vectors. In areas with AHS-infected animals, a higher incidence of disease occurs in low-lying, moist terrain and after periods of heavy rainfall. It is not known where the virus persisted over the winter during the outbreaks in the Near East and Spain. Transovarial transmission has not been recognized to occur in *Culicoides*.

If an infected animal is introduced into an AHS-free area during the time of the year when *Culicoides* are present and they have an opportunity to feed on the animal, an outbreak may occur. Because the horse is an amplifier of AHSV and the source of virus for arthropods, infected horses should be killed or placed in insect-proof isolation.

Generally, *Culicoides* move only a few miles from their breeding site. However, they can be transported long distances by wind. AHS probably got to Cyprus by wind-borne *Culicoides*.

An infected horse may have sufficient virus in the blood to infect vectors for up to 12 days after infection. Viremia may last up to 40 days.

Zebras and donkeys have a higher viremia for a longer time than horses.

Horses that recover from an infection have a lifetime immunity to that infecting virus and may have partial immunity to other serotypes. Foals will acquire a passive immunity from immunized mares.

Incubation Period

11

In experiments with horses, AHS usually has a 5- to 7-day incubation period. In natural infections, circumstantial evidence indicates that the incubation period is from 7 to 14 days.

Pathogenesis

Horses develop a fever of 102 to 106 °F (38.9 to 41.1 °C), and a moderately high titer of virus is present in lymph nodes, spleen, lung, and blood. Only trace amounts of virus are present in serum, excretions, and secretions. The horse can have a high enough viremia during the febrile period (4 to 9 days) to infect feeding arthropods. Zebras and donkeys can be viremic for 28 days or longer.

AHSV has been found in endothelial cells of the lungs; thus the pulmonary edema may be due to endothelial cell damage.

Clinical Signs

12

The first sign of AHS is a fever of 102 to 106 °F (38.9 to 41.1 °C). The next sign that most commonly appears is congestion of the conjunctivae. The severity of the conjunctival congestion is a good indication of the severity of the infection.

After these initial signs of illness, the disease can progress in one of four forms. Experimentally, the pulmonary, cardiac, and mixed forms of AHS have occurred in horses inoculated with aliquots from one viral pool. Thus it seems that the form of the disease depends on an individual horse–virus interaction.

Pulmonary Form (peracute form)

13 Fever, which may reach a maximum of 104 to 106 °F (40 to 41.1 °C), is followed by increasingly more rapid respiration and abdominal expiration. The respiratory rate may reach 60 to 70 per minute. Coughing and sweating commonly occur. The animal may also appear colicky—getting up and down and rolling. As pulmonary distress increases, the animal stands with its forelegs apart, head extended, and nostrils dilated. Once foam appears in the nostrils, death follows rapidly. Even during the terminal stages of the disease, the animal may drink and eat.

Cardiac Form (subacute edematous form)

14 The incubation period is usually longer (7 to 14 days) than in the pulmonary form. Fever of 102 to 106 °F (38.9 to 41.1 °C) usually lasts for 3 to 6 days. At the end of the febrile period, marked swelling of the head and neck may occur. Classic areas for subcutaneous swelling to occur are the supraorbital fossa and conjunctiva, and then the lips, cheeks, tongue, intermandibular space, laryngeal area, neck, brisket, and ventral thorax. In contrast to other equine diseases in which there is subcutaneous edema, no edema of the lower parts of the legs occurs in AHS. There may be petechial hemorrhages on the ventral surface of the tongue and in the conjunctiva. As the edema progresses, there may be restlessness and signs of abdominal pain and pulmonary edema. Finally the animal becomes prostrate and dies. Again, even during the terminal stages of the disease, the animal may drink and eat.

If the disease is not fatal, the edema gradually subsides over 3 to 8 days.

Mixed Form (acute)

15 The clinical signs are a mixture of pulmonary and cardiac forms; the signs of one form may predominate. The mixed form is the type of AHS more frequently seen at necropsy.

Mild Form (AHS fever)

16 This is the mildest form of AHS and can be subclinical or inapparent. This form occurs in zebras, donkeys, and horses with a heterologous immunity. Fever is usually intermittent—the animal has a normal temperature in the morning but is febrile in the afternoon. The fever may reach 104 °F (40 °C) for 1 or 2 days. Other clinical signs, such as an increased respiratory rate, mild conjunctival congestion, accelerated pulse, and some loss of appetite are rare, but when they do occur are mild. After 1 or 2 days, there is rapid recovery.

Photographs of Clinical Signs

17 Depressed horse.

18 Depressed horse. There is a small amount of nasal exudate and swelling of the supraorbital fossa.

19 Swelling of the supraorbital fossa.

20 Severe congestion and some edema of the conjunctiva.

Gross Lesions

21 The predominant gross lesions will vary with the form of the disease. The veterinarian should begin a necropsy with a careful reflection of the skin over the head, neck, shoulder, and brisket areas, looking for subcutaneous edema. Next, he or she should carefully examine the intermuscular fascia in the neck and the fascia along the ligamentum nuchae and the jugular furrow for edema. In some cases of AHS, edema of the intermuscular fascia in the neck may be the only lesion. If the edema in the neck is severe, there may be edema of the intermuscular fascia along the back and in the thighs. This edema is characteristic of the cardiac form of AHS.

Respiratory System

22. In the pulmonary form of AHS, hydrothorax (several hundred milliliters to several liters) is quite common. The lungs remain fully distended when the thoracic cavity is opened and are very heavy when removed. The subpleural area and the interlobular septa are greatly distended by fluid. The bronchi, trachea, pharynx, and nostril may contain foam if the carcass is fresh.

In the cardiac form, the lungs may be essentially normal or may have some congestion due to cardiac failure.

Cardiovascular System

23 In the cardiac form of AHS, heart lesions and lesions of cardiac failure are the predominant findings—massive hydropericardium and petechial and ecchymotic hemorrhages in the epicardium and endocardium. There is frequently myocardial necrosis in the papillary muscles.

Gastrointestinal Tract

24 There may be petechial hemorrhages on the ventral surface of the tongue.

There may be mild to severe petechial hemorrhages on the parietal and visceral peritoneum (a nonspecific lesion for viremia or septicemia).

There may be congestion of the stomach and liver and edema of mesenteric lymph nodes.

Photographs of Gross Lesions

25 To detect edema in AHS, reflect the skin as shown. Edema can be seen in a cut through the superficial neck muscle behind the ear.

26 The neck muscles should be cut and separated and the intermuscular fascia should be examined for edema.

27 An example of severe edema of intermuscular fascia.

28 Another example of severe edema of intermuscular fascia.

29 Horse dead of severe pulmonary edema.

30 Lung from a horse with severe pulmonary edema. The lung is fully distended, interlobular septa are widened by edema, and subpleural edema is present along the ventral edge of the lung.

31 Another lung with less severe pulmonary edema.

32 Petechiae on the ventral surface of the tongue.

33 Petechial hemorrhages in the visceral peritoneum.

34 Severe congestion of vessels in the mesentery and a few petechiae.

35 Petechial hemorrhages on the diaphragm.

36 Severe hydropericardium.

37 Necrosis in the papillary muscle.

Microscopic Lesions

38 There is no pathognomonic lesion for AHS; for a diagnosis, AHS has to be suspected clinically or at necropsy. Histologic lesions in the lung will be edema and some perivascular lymphocytic infiltration. Cardiac lesions will consist of hemorrhage, myocarditis, and focal myocardial necrosis. Spleen and lymph nodes will have some depletion of lymphocytes.

39 Photomicrograph of lung with pulmonary edema. Note the widely distended interlobular septum and distended alveoli.

Morbidity and Mortality

40 Morbidity associated with AHS depends on the exposure to infected *Culicoides*. Mortality will vary with serotype and strain of AHSV, the host infected, and immunity. Mortality in naive horses can be high.

Diagnosis

Field Diagnosis

41 It will be difficult to suspect AHS during the early febrile stage of disease. Suspect AHS if, during the season of the year when there are insect vectors, horses develop some of the following signs: fever, dyspnea, edema of the supraorbital fossa, subcutaneous edema of the head and/or neck, pulmonary edema, or death. If some of the above signs are observed, particular attention should be given to looking for AHS lesions at necropsy.

Specimens for Laboratory

42 Refrigerate but do not freeze the following specimens and send them to the laboratory:

- Blood, preferably with heparin as an anticoagulant (other anticoagulants can be used), or blood in an equal volume of OCG
- Pieces of spleen, mediastinal and mesenteric lymph nodes, lung, and liver
- At least 5 mL of serum from acute and convalescent animals

In addition, send the following:

- Blood smears (at least six) fixed in absolute methanol
- Tissues in 10-percent formalin, from spleen, liver, lung, kidney, heart, lymph nodes, and brain

Laboratory Diagnosis

Virus Isolation

43 Confirmation of an initial case of AHS in an area normally free of the disease requires isolation and identification of the virus. AHSV can be isolated from heparinized blood, spleen, lymph node, or lung collected at necropsy using cell culture (BHK21 or Vero cells), intracerebral inoculation of mice that are 2 to 3 days old, or intravenous inoculation of embryonated eggs at day 10 to

12. We recommend that more than one system of virus isolation be used. The incubation period in mice can be 4 to 20 days; then the mice die. Viral isolates are identified by group-specific tests such as complement fixation, enzyme-linked immunosorbent assay (ELISA), or immunofluorescence. Determination of the serotype is done by plaque reduction or plaque inhibition using known antisera.

Serology

44 The antibody to AHS can be detected starting about 10 days after infection. Group-specific tests are complement fixation (CF antibody present 4 to 6 months), immunofluorescent assay (IFA), ELISA, and immunodiffusion. Detectable antibodies are present for 1 to 4 years after infection.

Vaccination

45 The first vaccine used was attenuated by adapting the virus to grow in an adult mouse brain. This neurotropic vaccine would occasionally cause encephalitis in a horse. A plaque was selected from this vaccine and propagated in Vero cells. This Vero cell vaccine is the currently used vaccine. In South Africa, veterinarians use two quadrivalent vaccines administered 3 weeks apart. Outside the area where AHS is endemic, a monovalent vaccine corresponding to the type in the outbreak can be used.

In an experiment with serotype 4 live viral vaccine, the vaccine protected horses challenge-inoculated with a virulent serotype 4 AHSV against clinical disease but did not prevent infection and viremia. Therefore, at least with this serotype, a vaccinated horse could be an amplifier of AHSV.

Recently, a major biological firm produced a serotype 4 inactivated vaccine. Two inoculations of this vaccine gave better protection against challenge inoculation than the attenuated type 4 viral vaccine.

Control or Eradication

46

- Place animals inside from dusk until dawn.
- Use insect control and repellants.
- Quarantine horses from an infected area for 60 days in an insect-free area.
- Stop movement of animals.
- Knowing that the disease is vector transmitted, establish an appropriate area quarantine.
- Take temperature of animals at least daily and kill febrile animals or place them in an insect-free area.
- Vaccinate.